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ORIGINAL CONTRIBUTION

Pulse consumption improves indices of glycemic control in adults with and without type 2 diabetes: a systematic review and meta-analysis of acute and long-term randomized controlled trials

Maryam S. Hafiz[1,2] · Matthew D. Campbell[3,4,5] · Lauren L. O'Mahoney[6] · Melvin Holmes[1] · Caroline Orfila[1] · Christine Boesch[1]

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Abstract

Purpose Findings from randomized controlled trials (RCTs) evaluating the effect of pulse intake on glycemic control are inconsistent and conclusive evidence is lacking. The aim of this study was to systematically review the impact of pulse consumption on post-prandial and long-term glycemic control in adults with and without type 2 diabetes (T2D).

Methods Databases were searched for RCTs, reporting outcomes of post-prandial and long-term interventions with different pulse types on parameters of glycemic control in normoglycemic and T2D adults. Effect size (ES) was calculated using random effect model and meta-regression was conducted to assess the impact of various moderator variables such as pulse type, form, dose, and study duration on ES.

Results From 3334 RCTs identified, 65 studies were eligible for inclusion involving 2102 individuals. In acute RCTs, pulse intake significantly reduced peak post-prandial glucose concentration in participants with T2D (ES −2.90; 95%CI −4.60, −1.21; $p \leq 0.001$; $I^2 = 93\%$) and without T2D (ES −1.38; 95%CI −1.78, −0.99; $p \leq 0.001$; $I^2 = 86\%$). Incorporating pulse consumption into long-term eating patterns significantly attenuated fasting glucose in normoglycemic adults (ES −0.06; 95%CI −0.12, 0.00; $p \leq 0.05$; $I^2 = 30\%$). Whereas, in T2D participants, pulse intake significantly lowered fasting glucose (ES −0.54; 95%CI −0.83, −0.24; $p \leq 0.001$; $I^2 = 78\%$), glycated hemoglobin A1c (HbA_{1c}) (ES −0.17; 95%CI −0.33, 0.00; $p \leq 0.05$; $I^2 = 78$) and homeostatic model assessment of insulin resistance (HOMA-IR) (ES −0.47; 95%CI −1.25, −0.31; $p \leq 0.05$; $I^2 = 79\%$).

Conclusion Pulse consumption significantly reduced acute post-prandial glucose concentration > 1 mmol/L in normoglycemic adults and > 2.5 mmol/L in those with T2D, and improved a range of long-term glycemic control parameters in adults with and without T2D.

PROSPERO registry number (CRD42019162322).

Keywords Pulses · Glucose · Diabetes · Postprandial glycemia · Systematic review · Meta-analysis

✉ Christine Boesch
c.bosch@leeds.ac.uk

[1] School of Food Science and Nutrition, University of Leeds, Leeds LS2 9JT, UK

[2] Faculty of Applied Medical Sciences, Department of Clinical Nutrition, King Abdul-Aziz University, Jeddah, Saudi Arabia

[3] School of Nursing and Health Sciences, Faculty of Health Sciences and Wellbeing, University of Sunderland, Sunderland, UK

[4] Wellcome-MRC Institute of Metabolic Science, University of Cambridge, Cambridge, UK

[5] Leeds Institute of Cardiovascular and Metabolic Medicine, University of Leeds, Leeds, UK

[6] Leicester Diabetes Centre, University of Leicester, Leicester, UK

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Abbreviations

ADA	American diabetes association
AUC	Area under the curve
CHO	Carbohydrates
CI	Confidence interval
EASD	European association for the study of diabetes
ES	Effect size
GI	Glycemic index
HbA_{1c}	Glycated hemoglobin A1c
HOMA-IR	Homeostatic model assessment of insulin resistance
PPGR	Post-prandial glucose response
PI	Prediction intervals
RCTs	Randomized controlled trials
SCFA	Short-chain fatty acids
T2D	Type 2 diabetes

Introduction

The European Association for the Study of Diabetes (EASD) and the American Diabetes Association (ADA) advocate increasing fiber intake, specifically through the consumption of pulses as a means to improve blood glucose control in adults with and without T2D [1, 2]. Several epidemiological studies have reported inverse associations between pulse intake and incidence of T2D [3, 4]. In addition, RCTs suggest that pulse consumption may improve acute post-prandial glucose control, and lower fasting blood glucose, insulin and HbA_{1c} levels when incorporated into long-term eating patterns [5, 6].

Pulses are rich sources of low glycemic index (GI) carbohydrates (CHO, up to 65%), and protein with up to 25% (dry weight) [7]. Low GI, fiber-rich foods have been shown to reduce post-prandial glycemic responses (PPGR) compared to foods with similar CHO content [8, 9], as well as protein addition to breakfast is suggested to improve PPGR [10]. In addition, pulses contain phytochemicals such as catechins and procyanidins which have been demonstrated to suppress the enzymatic activity of CHO digestive enzymes including α-amylase and α-glucosidase thereby contributing towards improved post-prandial glycemic control [11–13].

A number of randomized controlled trials have assessed the effect of pulse intake on acute post-prandial and long-term glucose response [14–23]. The studies differed in the type of pulses used, processing, doses and control group, and in different volunteer profiles [6, 24–33]. The study outcomes vary considerably with low quality of evidence and, therefore, the true effect size of pulse intake on measures of glycemic handling remains unclear [34]. A previous systematic review and meta-analysis by Sievenpiper et al. (2009) concluded a significant reduction in fasting blood glucose and insulin after long-term consumption of pulses alone, as part of low GI or high-fiber diets [35]. However, the review was published in 2009 and only long-term trials were included in their review. Considering that there are more than 20 long-term trials published since 2009 and given the lack of summarized evidence on post-prandial glucose response after intake of pulses, the aim of the current systematic review is to update the evidence on long-term effects of pulse consumption on glycemic indices as well as integrate the acute glucose response along from RCTs on individuals with and without T2D.

Methods

The guidelines of Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [36] were followed for conducting this systematic review and meta-analysis. The systematic review was prospectively registered with PROSPERO (CRD42019162322).

Search strategy and study selection

We searched Pubmed and Cochrane library databases to identify all randomized clinical trials (RCTs) conducted and relevant to the topic until 28th of January 2021. Full search terms are illustrated in Supplemental Table 1. No filters for language, date of publication, or design of the study were applied when searching the databases. An additional manual search was conducted through reviewing reference lists of selected articles and reviews.

The study selection process was performed in duplicate independently by two reviewers by initially reviewing the titles and abstracts and finally reviewing the full texts to identify all eligible RCTs. Included studies were randomized controlled trials either acute (assessing single meal response) or long-term (assessing intake > 2 weeks) [37], including all adults except type 1 diabetes mellitus and gestational diabetes, investigating the effect of intake of pulses in comparison to control diet, on parameters of glycemic control measured using capillary or venous blood. Studies were excluded if they investigated legumes other than pulses such as soya beans or green peas, failed to use a matched available carbohydrate control in acute glucose response trials; the pattern of pulse consumption was not specified; used pulse fractions such as their extracts; protein isolates or husk only; reported subsequent second meal effect rather than immediate response; did not exclude or account for confounding factors whether in participants or intervention diets that might impact glucose metabolism; or outcome measures of glycemic control were not reported. In studies where different interventions were used in different arms, only data from arms that met the eligibility criteria were included in

the analysis. Included trials were limited to published and peer-reviewed RCTs available as full texts in English. Corresponding authors were contacted to request the full text in cases where the full text was not available online before deciding on exclusion.

Data extraction and quality assessment

Data were extracted by single author and included: first author and year of publication; publishing journal; design of the study; intervention arms; number of visits in acute studies; study duration in long-term studies; sample size and participant characteristics (gender, health status, age group and body mass index); intervention design and control (type, dose and format); pulse characteristics (type, dose and physical form). The outcome measures of acute trials were extracted for means and standard deviations of baseline and post-prandial glucose (mmol/L) and insulin (mIU/L) values and their area under the curves (AUCs). In the long-term trials, baseline and post-intervention mean and standard deviation values were extracted for fasting blood glucose (mmol/L), insulin (mIU/L), glycated hemoglobin (%) and insulin resistance expressed as HOMA-IR. Where data were presented in non-standard units, they were converted to standard reporting units. If data were available in figure format only, values were digitized using Graph Digitizer. In trials not reporting the standard deviation, the values were derived from standard errors or confidence intervals (CI).

Bias assessment of individual trials was performed independently by two reviewers following the updated Cochrane Collaboration's tool for assessing risk of bias (RoB2) [38]. The trials were classified into three categories "high risk, low risk, or some concerns raised" in five domains which are as follows: randomization process, deviations from intended interventions, missing outcome data, measurement of the outcome, and selection of the reported results. The proposed algorithm was followed in signaling questions to judge risk of bias of each domain as well as overall risk of bias. Publication bias was visually assessed by inspection of funnel plots and quantitatively using Egger's test for each outcome [39].

Data analysis

Data were analyzed using Review Manager (RevMan) 5.3.5 Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014; and R Core Team (2020), R: A language and environment for statistical computing, R foundation for statistical computing, Vienna, Austria. The random effects model was chosen assuming that the RCTs included in the analysis were functionally inequivalent. Weighted averages were calculated in trials using more than one arm for intervention to avoid errors in analyses [40]. RCTs not reporting the amount of pulses administered were excluded from the meta-analysis. Pooled random effects analyses were performed to estimate the effect size in acute and long-term RCTs on normoglycemic and T2D participants. The entered data included sample size, reported means and standard deviations for intervention arms and their matched carbohydrate controls of each trial. Effect size was estimated for post-prandial glucose and insulin response in acute RCTs and for the difference between pre- and post-intervention in fasting blood glucose, insulin, glycated hemoglobin, and HOMA-IR values as raw mean differences and 95% CIs. A negative ES was interpreted as favoring pulse intake, while a positive ES favored control. The inter-study variance was assessed using tau^2 and I^2 along with calculation of prediction intervals (PI). Sensitivity analysis was performed to explore the impact of removing one RCT on outcomes, as well as investigate removal of studies with high risk of bias on ES [41].

Subgroup analysis and meta-regression were performed if ≥ 10 RCTs could be included in the meta-analysis to explore the variations in ES, considering pulse type or processing method used in intervention arms, control food used for comparison, and dose or duration of the study as variables [41].

Grading the evidence

The Grading of Recommendations Assessment, Development and Evaluation (GRADE) tool was conducted by single author for interpreting outcome data to evaluate the certainty of evidence [42]. Evidences on the ES can be graded to 'very low', 'low', 'moderate', or 'high' based on evaluation outcomes in five domains. The domains are as follows: overall risk of bias, inconsistency, indirectness, imprecision, and other considerations.

Results

A total of 3334 studies were identified through database searches and additional sources, of which 2966 were screened based on title and abstract only. Of these, 150 studies were reviewed as full text and subsequently 85 studies were excluded for not meeting the inclusion criteria, as detailed in the study selection flowchart (Fig. 1). In total, 65 RCTs were included in the final systematic review and 59 RCTs in the meta-analysis, involving a total of 2102 individuals (905 with and 1197 without T2D). The RCTs were classified according to the design of the study as acute post-prandial ($n = 37$, Tables 1, 2) or long-term ($n = 28$, Tables 3, 4) trials and separated into normoglycemic (Tables 1, 3) and T2DM (Tables 2, 4).

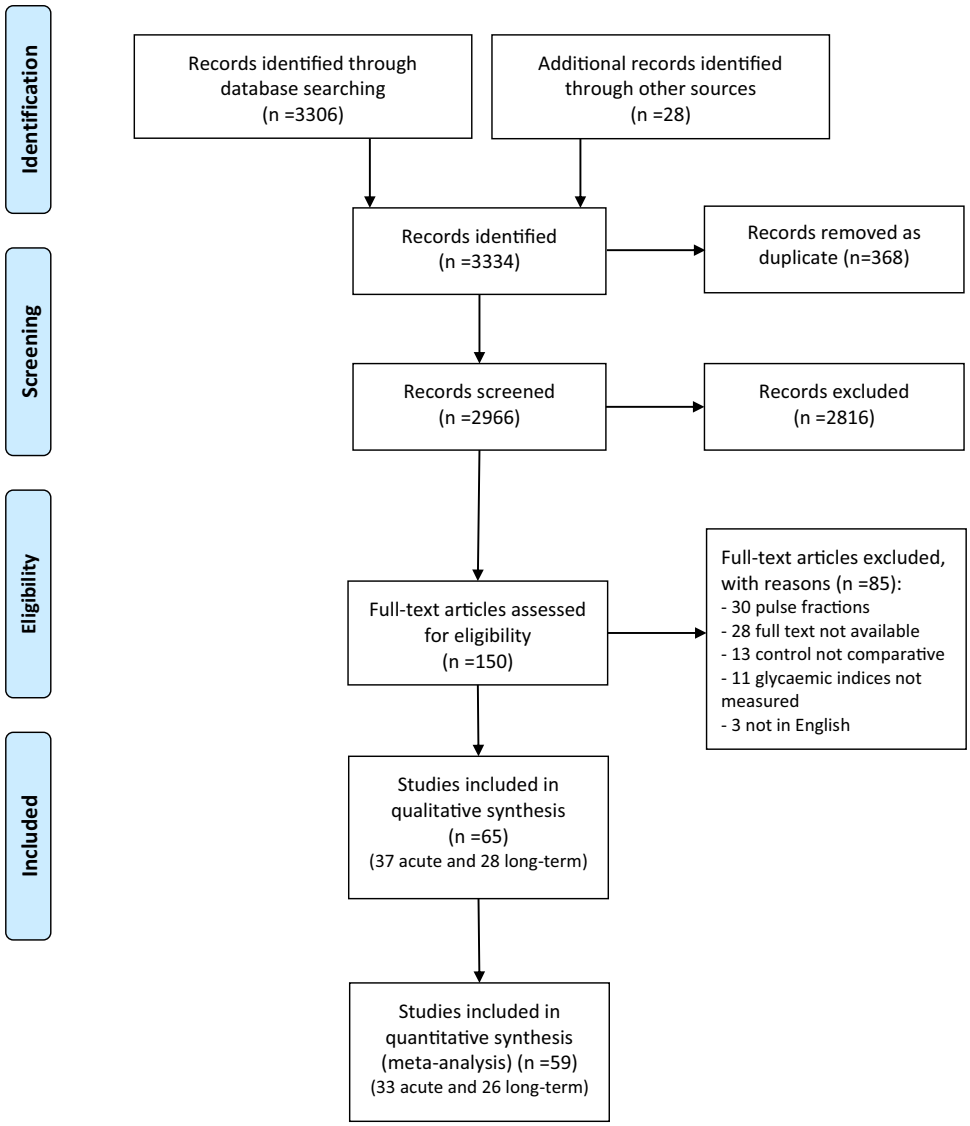

Fig. 1 Flow diagram of trial selection

Assessment of risk of bias across the studies indicated concerns for the majority of RCTs due to lack of information on randomization concealment as well as selection of the reported results (Supplemental Table 2). There were ten RCTs that fell into the 'high risk' category due to concerns in three or more domains. These were mainly the trials that were published more than 20 years ago; in appreciation of the fact that the standards on reporting RCTs were substantially different then, we have not removed these studies from the meta-analysis.

Parameters of post-prandial glycemic control

The meta-analysis showed that pulse intake significantly improved parameters of post-prandial glycemic handling. Post-prandial plasma glucose was overall significantly reduced in normoglycemic adults ($n = 27$ RCTs, ES −1.38; 95% CI −1.78, −0.99; $p \leq 0.001$; $I^2 = 86\%$, PI −3.33, 0.57) and in adults with T2D ($n = 6$ RCTs, ES −2.90; 95% CI −4.60, −1.21; $p \leq 0.001$; $I^2 = 93\%$, PI −8.97, 3.17) (Figs. 2, 3), with high heterogeneity between studies. Egger's test of publication bias did not indicate presence of funnel plot asymmetry ($p > 0.05$) (Supplemental Fig. 1). Subgroup analysis of pulse type revealed that lentils ($n = 9$ RCTs) were most effective in reducing PPGR (ES −1.60; 95% CI −2.23, −0.97; $p \leq 0.0001$, $I^2 = 84\%$), followed by dried peas ($n = 5$ RCTs; ES −1.32; 95% CI −2.07, −0.56; $p \leq 0.005$, $I^2 = 81\%$), beans ($n = 14$ RCTs; ES −1.18; 95% CI −1.74, −0.62; $p < 0.0001$, $I^2 = 82\%$), and chickpeas ($n = 11$ RCTs; ES −0.97; 95% CI −1.48, −0.47; $p < 0.001$, $I^2 = 78\%$). However, the differences in ES were not significant between types of pulses ($p = 0.49$) (Supplemental Fig. 2). Furthermore, analysis and meta-regression of processing method revealed that ES was significantly

Table 1 Summary of acute RCTs investigating the effect of pulse intake on glycemic indices in normoglycemic adults

References	Country of study	Design	N	Age, y[1]	BMI[1]	Pulse type	Format	Other CHO source	Total CHO (pulse only), g	Control	Outcomes
Agustia et al. [43]	Indonesia	NR	11	20.1 ± 1.3	20.9 ± 1.9	Beans	Flour	Rice	50	Glucose	Glucose
Akhtar et al. [44]	Pakistan	C	24	22.5 ± 2.4	21.8 ± 1.7	Beans	Flour	Wheat flour	50 (20)	Wheat flour	Glucose, insulin
Anderson et al. [18]	Canada	C	17	22.1 ± 3.0	22.9 ± 1.2	Beans, lentils, chickpeas	Whole, pureed and Flour	Tomato sauce	38.7 (25)	Whole wheat flour	Glucose
Anguah et al. [20]	US	C	12	28.0 ± 10.0	23.3 ± 3.1	Lentil	Whole, pureed	Rice, wheat	NR	Rice and egg burritos	Glucose
Augustin et al. [21]	Canada	C	10	53.0 ± 7.0	29.4 ± 3.8	Chickpeas	Pureed	–	25	White bread	Glucose, insulin
Boers et al. [23]	UK	C	12	37 ± 9	22.8 ± 1.6	Chickpeas	Flour	Wheat	57 (8.5)	High-fiber flat bread	Glucose
Bornet et al. [25]	France	C	6	23.9 ± 1.7	20.6 ± 1.7	Beans	Flour	–	35	Extruded wheat	Glucose, insulin
Dandachy et al. [27]	Lebanon	C	16	22.9 ± 12	22.7 ± 10.6	Chickpeas	Flour	Wheat	NR	Wheat flour	Glucose
Dilwari et al. [29]	India	C	6	36.3 ± 9.7	NR	Lentils, beans	Whole	–	50	Rice	Glucose
Greffeuille et al. [45]	France	C	15	24 ± 11.2	22.4 ± 7.0	Beans	Flour	Wheat	50 (17.5)	Wheat pasta	Glucose, insulin
Jenkins et al. [33]	UK	C	10	NR	NR	Beans; peas; chickpeas; lentils	Whole	–	50	White bread	Glucose
Jenkins et al. [30]	UK	C	9	29.0 ± 8.0	NR	Lentils	Whole	–	50	White bread	Glucose
Johnson et al. [46]	Australia	C	11	32.0 ± 6.6	24.7 ± 2.7	Chickpeas	Flour	Jam, milk	50 (NR)	White bread	Glucose, insulin
Marinangeli et al. [47]	Canada	C	22	NR	NR	Peas	Flour, whole	Wheat	50 (NR)	White bread	Glucose
Mehio et al. [48]	Lebanon	C	12	24.0 ± 3.4	22.8 ± 2.1	Chickpeas	Pureed	NR	50	White bread	Glucose, insulin
Mollard et al. [49]	Canada	C	25	21.3 ± 2.5	21.6 ± 1.5	Chickpeas, lentils, peas	Whole	Macaroni	98.7 (40)	Macaroni and cheese	Glucose
Moravek et al. [50]	Canada	C	24	27.4 ± 1.2	24.3 ± 0.5	Lentils	Whole	Rice/potato	50 (NR)	Rice or potatoes	Glucose, insulin
Nestel et al. [51]	Australia	C	19	61.5 ± 6.4	26.5 ± 3.8	Chickpeas	Pureed	Milk	50 (33)	White bread and jam	Glucose
Potter et al. [52]	US	C	8	NR	NR	Beans	Pureed	–	75	Brown rice	Glucose
Ramdath et al. [53]	Canada	C	10	45.1 ± 11.0	27.7 ± 6.1	Lentils	Whole	–	25, 25	White bread	Glucose
Ramdath, et al. [6]	Canada	C	10	40.0 ± 10.0	25.0 ± 4.1	Lentils	Whole, pureed and Flour	–	50	Potatoes	Glucose
Reverri et al., (2015) [54]	US	C	12	49.0 ± 14.0	32.2 ± 5.7	Beans	Pureed	–	NR	Couscous	Glucose
Tappy et al. [55]	Switzerland	C	6	NR	NR	Beans	Flakes	–	50	Potatoes	Glucose

Table 1 (continued)

References	Country of study	Design	N	Age, y[1]	BMI[1]	Pulse type	Format	Other CHO source	Total CHO (pulse only), g	Control	Outcomes
Torsdottir et al. [56]	Sweden	C	6	24.0 ± 6.0	22.2 ± 1.1	Beans	Pureed	–	43	Potatoes	Glucose, insulin
Traianedes et al. [57]	Australia	C	6	30.0 ± 10.0	24.3 ± 1.7	Beans	Whole	–	50	Glucose	Glucose, insulin
Winham et al. [58]	US	C	12	36.0 ± 15.0	23.3 ± 5.4	Black beans, chickpeas	Whole	Rice	50 (15)	Rice	Glucose, insulin
Wong et al. [59]	Canada	C	14	NR	NR	Beans, chickpeas, lentils, peas	Whole	–	50	White bread	Glucose
Yoshimoto et al. [60]	Japan	C	12	37.8 ± 9.5	22.9 ± 3.5	Peas	Flour	–	50	Rice	Glucose, insulin
Zafar et al. [61]	Kuwait	C	13	21.4 ± 2.3	23.6 ± 2.4	Chickpeas	Flour	Wheat, milk	NR	White bread	Glucose
Zhu et al. [62]	China	C	10	20.7 ± 2.3	22.0 ± 2.1	Beans	Whole	–	50	White rice	Glucose
Zurbau et al. [63]	Canada	C	21	26.7 ± 12.3	22.2 ± 2.8	Chickpeas	Whole	Tomatoes	50 (NR)	Potatoes	Glucose

[1]Age and BMI are reported as mean ± SD; *BMI* body mass index, *CHO* available carbohydrates, *C* crossover, *N* number of participants, *NR* not reported

Table 2 Summary of acute RCTs investigating the effect of pulse intake on glycemic indices in T2D adults

References	Country of study	Design	N	Age, y[1]	BMI[1]	Pulse type	Format	Other CHO source	Total CHO (pulse only), g	Control	Outcomes
Bornet et al. [24]	France	C	18	57 ± 8.5	27.9 ± 4.7	Lentils, beans	Whole	–	50	Glucose	Glucose, insulin
Jenkins et al. [31]	UK	C	6	43 ± 5	NR	Lentils	Whole	Soya	50 (23)	Whole meal bread	Glucose
Mani et al. [64]	India	C	6	58 ± 9	NR	Lentils	Whole	Semolina	50 (16)	Semolina	Glucose
Olmedilla-Alonso et al. [65]	Spain	C	12	66.4 ± 6.2	30.1 ± 3.6	Beans	Whole	–	57.8	White bread	Glucose, insulin
Schafer et al. [66]	Germany	C	9	61 ± 14	29.9 ± 8.7	Peas	Whole	Carrots	40 (37)	Potato	Glucose, insulin
Thompson et al. [67]	US	C	17	58.6 ± 20	31.9 ± 7.9	Beans	Whole	Rice	50 (15)	Rice only	Glucose

[1]Age and BMI are reported as mean ± SD; *BMI* body mass index, *CHO* available carbohydrates, *C* crossover, *N* number of participants, *NR* not reported

Table 3 Summary of long-term RCTs investigating the effect of pulse intake on glycemic indices in normoglycemic adults

References	Country of study	Design	Duration, weeks	N	Age, y[1]	BMI[1]	Intervention	Dose, g/day	Control	Outcomes
Abete et al. [14]	Spain	P	8	32	NR	32.5 ± 4.3	Low GI diet with pulse intake	130	Energy-restricted high GI diet	Glucose, insulin, HOMA-IR
Abete et al. [15]	Spain	P	8	35	38.0 ± 7.0	31.8 ± 3.0	High-pulse diet	100	Energy-restricted diet	Glucose
Abeysekara et al. [16]	Canada	C	8	87	59.7 ± 6.3	27.5 ± 4.5	Pulse-based diet	250	Regular diet	Glucose, insulin
Alizadeh et al. [17]	Iran	P	6	34	36.1 ± 8.2	NR	hypocaloric diet enriched in pulses	190	Hypocaloric diet	Glucose, insulin, HOMA-IR
Anderson et al. [19]	US	P	3	10	53.9 ± 8.5	NR	Beans supplemented diet	115	Oat bran diet	Glucose
Cryne et al. [26]	Canada	C	4	21	28.1 ± 5.9	25.2 ± 3.5	Spray-dried chickpeas, lentils, peas	100	Dehydrated potato flakes	Glucose, insulin, HOMA-IR
Gravel et al. [68]	Canada	P	16	132	51.7 ± 8.6	29.8 ± 5.1	Pulse-based meals	110	Isocaloric control meals	Glucose, insulin
Kim et al. [69]	Australia	C	4	51	35.1 ± 15.6	27.7 ± 6.9	Diet high in dairy, whole grains, nuts and pulses	150–225	Diet high in red and meat and refined grains	Glucose
Marinangeli et al. [70]	Canada	C	4	23	52.0 ± 11.2	30.5 ± 4.4	whole pea flour muffin	50	White wheat flour muffin	Glucose
Nestel et al. [51]	Australia	C	6	20	56.6 ± 7.6	25.6 ± 3.2	Chickpea based diet	200	Wheat-based diet	Glucose, insulin, HOMA-IR
Pittaway et al. [71]	Australia	C	5	27	50.6 ± 10.5	28.8 ± 4.4	Chickpeas based diet	200	Low fiber wheat-based diet	Glucose, insulin, HOMA-IR
Saraf-Bank et al. [72]	Iran	C	6	26	50.0 ± 6.6	28.9 ± 4.3	Habitual diet enriched with pulses	65	Habitual diet without pulses	Glucose, HbA_{1c}
Tonstad et al. [73]	US	P	16	123	48.4 ± 10.7	36.4 ± 3.5	High-fiber bean-rich diet	125	Low-carbohydrate diet	Glucose, HbA_{1c}
Tovar et al. [74]	Sweden	C	4	46	61.6 ± 5.4	28.8 ± 8.1	Whole grain, barley and pulse rich diet	168	Low pulse diet	Glucose, insulin, HbA_{1c}, HOMA-IR
Venn et al. [75]	New Zealand	P	72	113	42.0 ± 10.7	35.4 ± 5.5	High pulse diet	180	Low pulse diet	
Winham et al. [76]	US	C	8	16	43.0 ± 20.0	27.8 ± 5.6	Beans/peas enriched diet	120	Carrot enriched diet	Glucose, insulin, HbA_{1c}, HOMA-IR

[1]Age and BMI are reported as mean ± SD, *BMI* body mass index, *C* crossover, *N* number of participants, *NR* not reported, *P* parallel study design

Table 4 Summary of long-term RCTs investigating the effect of pulse intake on glycemic indices in T2D adults

References	Country of study	Design	Duration, weeks	N	Age, y[1]	BMI[1]	Intervention	Dose, g/day	Control	Outcomes
Hassanzadeh-Rostami et al. [77]	Iran	P	8	64	59.6 ± 5.9	27.3 ± 3.4	Pulses	100	Red meat	Glucose, insulin, HbA_{1c}
Hosseinpour-Niazi et al. [78]	Iran	C	8	31	58.1 ± 6.0	27.7 ± 3.3	Pulse-based TLC diet	190	Pulse-free TLC diet	Glucose, insulin
Islam et al. [79]	Bangladesh	P	4	30	52.4 ± 5.6	25.1 ± 2.2	Mixed pulse and wheat bread	NR	Wheat bread	Glucose
Jang et al. [80]	Republic of Korea	P	16	76	56.6 ± 8.6	24.6 ± 2.2	Black bean powder mixed with wholegrains powder	15	Cooked refined rice	Glucose, insulin, HOMA-IR
Jenkins et al., (2012) [81]	Canada	P	12	121	53.0 ± 10.0	29.9 ± 5.5	Low GI pulse diet	190	High wheat fiber diet	Glucose, HbA_{1c}
Jimenez-Cruz et al. [82]	US	C	6	14	53.0 ± 9.0	32.3 ± 5.9	Low GI Mexican style diet with pulses	35	High GI Mexican style diet	
Jimenez-Cruz et al. [83]	US	C	3	8	51.0 ± 3.0	30.7 ± 7.9	Low GI high fiber diet with pulse	NR	High GI low fiber diet	Glucose, HbA_{1c}
Kang et al. [84]	Republic of Korea	P	12	185	50.4 ± 9.9	25.5 ± 3.2	Whole grains and pulses	30–70	Refined rice diet	Glucose, insulin, HOMA-IR
Kim et al. [5]	Republic of Korea	P	12	99	55.4 ± 11.9	24.1 ± 3.4	Whole grains and pulses	30–70	Refined rice diet	Glucose, insulin, HbA_{1c}, HOMA-IR
Kim et al. [85]	Republic of Korea	P	12	80	NR	NR	Whole grains and pulses	30–70	Refined rice diet	Glucose, insulin, HbA_{1c}, HOMA-IR
Liu et al. [86]	China	P	4	106	57.4 ± 8.8	26.6 ± 1.0	Extruded adzuki bean convenient food	170	Low GI diet	Glucose, insulin, HbA_{1c}
Winham et al. [87]	US	C	8	23	45.9 ± 21	27.4 ± 5.1	Canned baked navy beans	130	Canned carrots	Glucose, insulin, HbA_{1c}, HOMA-IR

[1]Age and BMI are reported as mean ± SD; *BMI* body mass index, *C* crossover, *N* number of participants, *NR* not reported

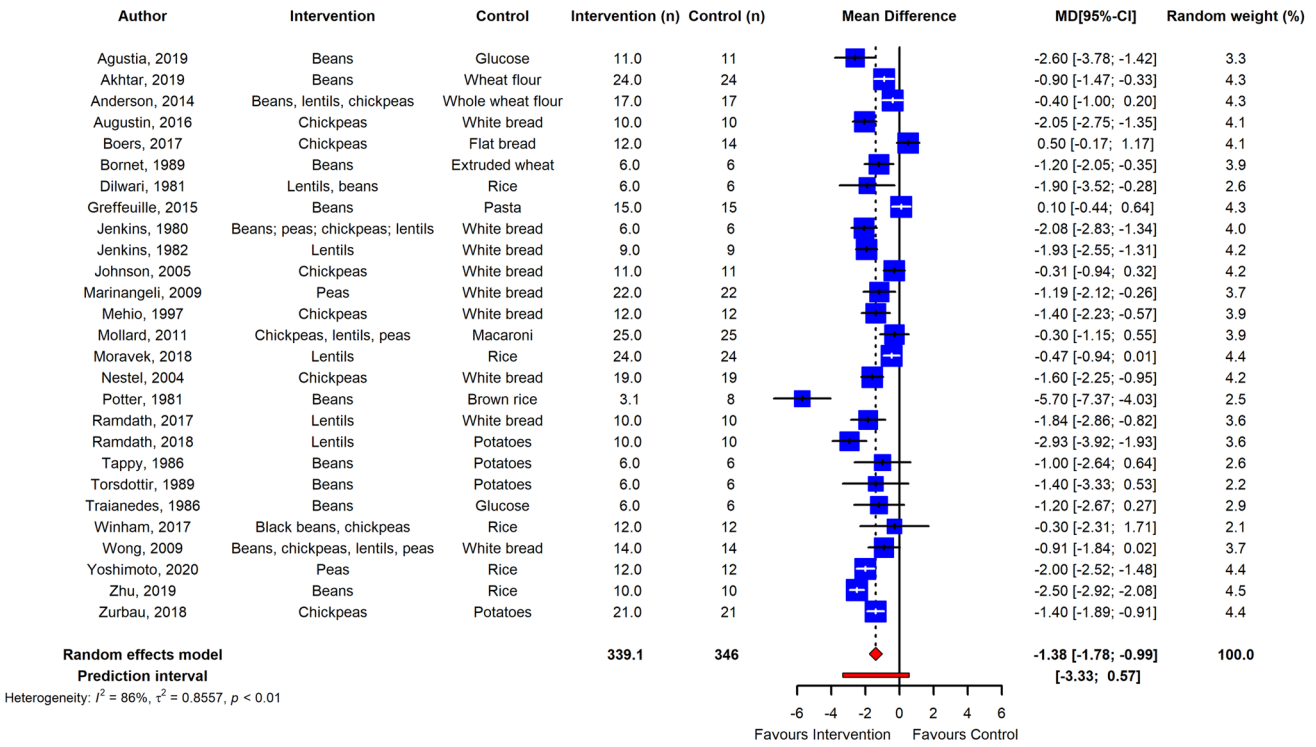

Fig. 2 Pooled effect using inverse-variance random effect model (mean difference and 95% CI) of acute trials investigating pulse intake on post-prandial glucose response among healthy individuals. The effect size was statistically significant for normoglycemic adults

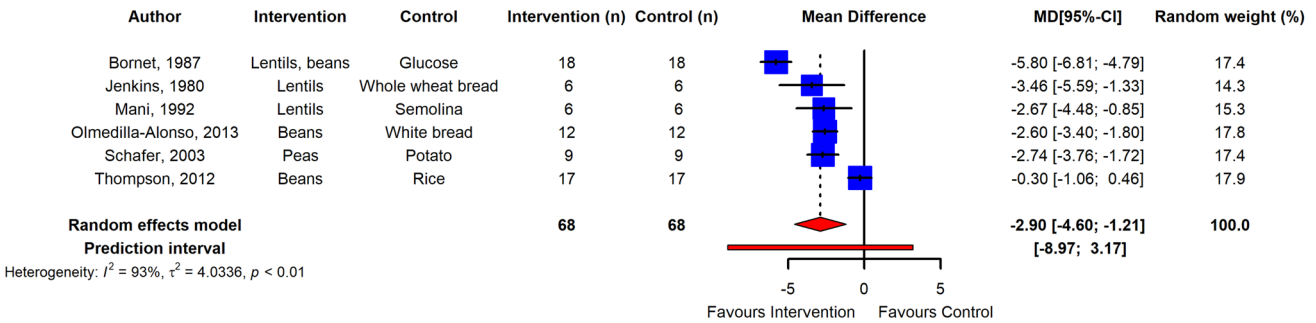

Fig. 3 Pooled effect using inverse-variance random effect model (mean difference and 95% CI) of acute trials investigating pulse intake on post-prandial glucose response among T2D individuals. The effect size was statistically significant for adults with T2D

lower when pulse flour was used as intervention ($n = 10$ RCTs; ES −0.81; 95% CI −1.33, −0.29; $p \leq 0.005$, $I^2 = 83\%$) compared to whole ($n = 14$ RCTs; ES −1.84; 95% CI −2.32, −1.37; $p \leq 0.0001$, $I^2 = 80\%$) and pureed pulse ($n = 7$ RCTs; ES −1.65; 95% CI −2.33, −0.98; $p \leq 0.0001$, $I^2 = 70\%$) with ($p < 0.05$) for subgroup differences (Supplemental Fig. 3). Moreover, subgroup analysis by grouping control foods used in the post-prandial trials suggested that the ES was greater when potatoes were used as control and pasta was the lowest (Supplemental Fig. 4). Sensitivity analysis by removal of studies with high risk of bias did not change the ES.

ES of post-prandial insulin responses were also significantly lower in both adults with and without T2DM ($n = 3$ RCTs; ES −19.43; 95% CI −24.01, −14.85; $p \leq 0.0001$, $I^2 = 0\%$) and ($n = 11$ RCTs; ES −11.26; 95% CI −22.11, −0.41; $p \leq 0.05$, $I^2 = 90\%$), respectively.

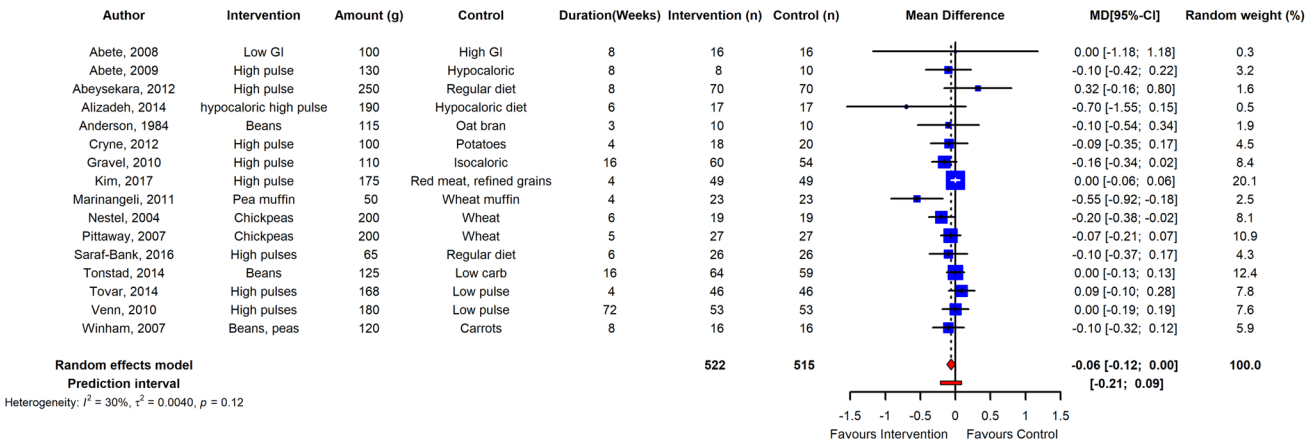

Fig. 4 Pooled effect using inverse-variance random effect model (mean difference and 95% CI) of long-term trials investigating pulse intake on fasting glucose among healthy individuals. The meta-analysis concluded that long-term pulse intake has small but significant effect on reducing fasting blood glucose levels in normoglycemic adults

Long-term parameters of glycemic control

The meta-analysis revealed that long-term pulse intake has a small reducing effect on fasting blood glucose levels in normoglycemic adults (n = 16 RCTs) with low heterogeneity between studies (ES −0.06; 95% CI −0.12, 0.00; $p \leq 0.05$; $I^2 = 30\%$; PI −0.21, 0.09) (Fig. 4). Sensitivity analysis showed that independent removal of one trial changed the ES interpretation from significant to non-significant. Pulse consumption in normoglycemic adults had no significant effect on fasting insulin, HbA_{1c} and HOMA-IR, although the effect direction was toward reduction ($n = 9$ RCTs; ES −0.11; 95% CI −0.76, 0.55; $p = 0.75$); ($n = 4$ RCTs; ES −0.03; 95% CI −0.11, 0.06; $p = 0.54$); ($n = 7$ RCTs; ES −0.02; 95% CI −0.18, 0.14; $p = 0.78$), respectively.

Long-term pulse intake resulted in a significant reduction of fasting blood glucose in adults with T2D as estimated from data of 10 RCTs (ES −0.54; 95% CI −0.83, −0.24; $p \leq 0.005$; $I^2 = 78\%$; PI −1.44, 0.37), albeit with high heterogeneity among studies (Fig. 5). HbA_{1c} and HOMA-IR were also significantly reduced in adults with T2DM with high heterogeneity between studies ($n = 6$ RCTs; ES −0.17; 95% CI −0.33, −0.00; $p \leq 0.05$; $I^2 = 78$; PI −0.69, 0.36) and ($n = 4$ RCTs; ES −0.47; 95% CI −1.25, −0.31; $p \leq 0.05$; $I^2 = 79\%$; PI −3.63, 2.69) (Fig. 6). Sensitivity analysis revealed that independent removal of one trial in estimation of ES of HbA_{1c} reduced the heterogeneity significantly [77], and removal of two RCTs changed the interpretation from significant to non-significant when estimating the ES of HOMA-IR [5, 85]. However, reduction in fasting blood insulin in T2DM adults was not significant ($n = 8$ RCTs, ES −1.18; 95% CI −2.54, −0.08; $p > 0.05$; $I^2 = 63\%$). Egger's test did not indicate funnel plot asymmetry in long-term trials ($p > 0.05$) (Supplemental Figs. 5, 6).

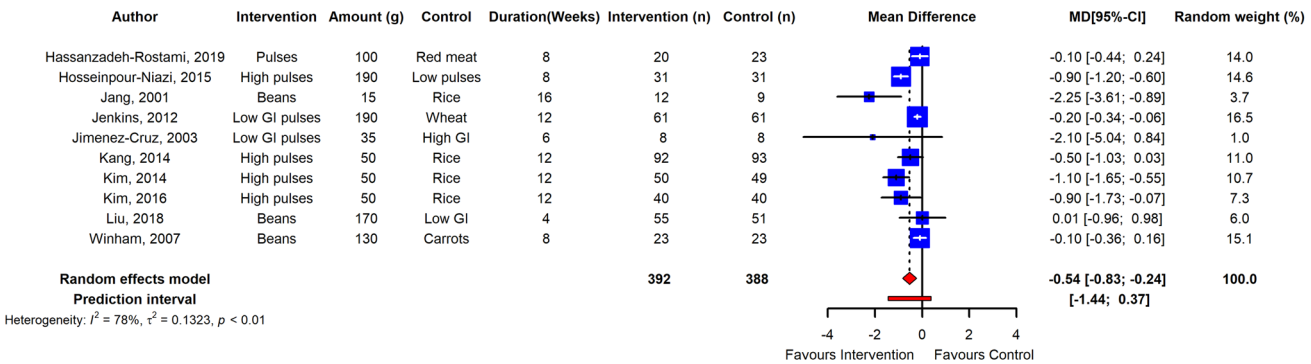

Fig. 5 Pooled effect using inverse-variance random effect model (mean difference and 95% CI) of long-term trials investigating pulse intake on fasting glucose among T2D individuals. Long-term pulse intake resulted in a significant reduction of fasting blood glucose in adults with T2D

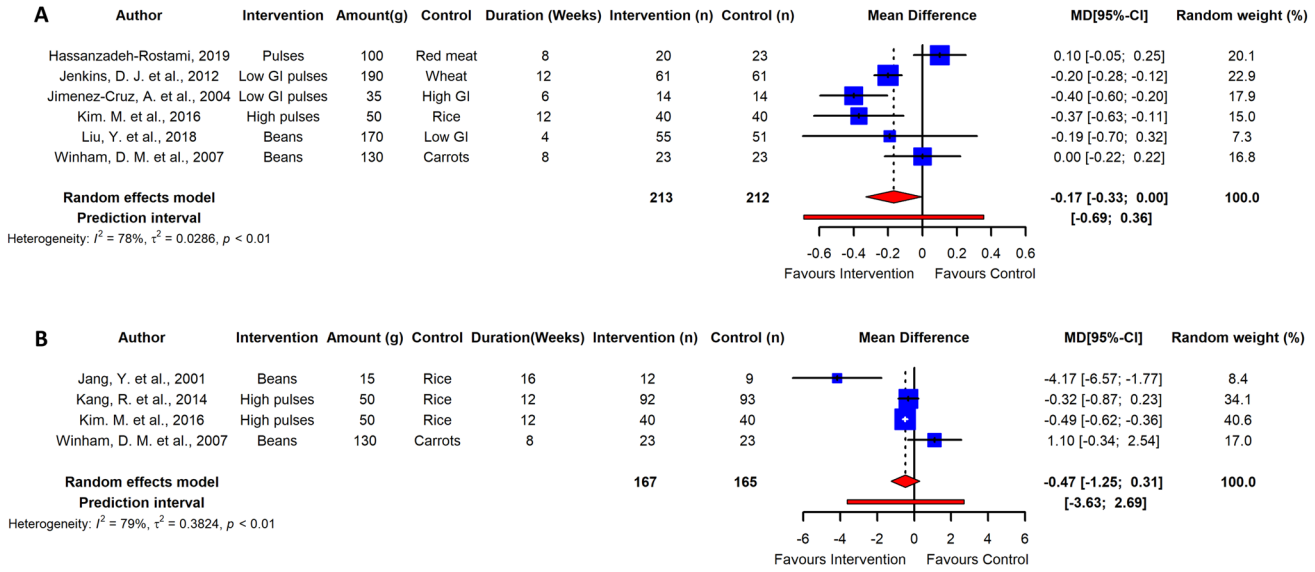

Fig. 6 Pooled effect using inverse-variance random effect model (mean difference and 95% CI) of long-term trials investigating pulse intake on fasting glycated hemoglobin (**a**); and HOMA-IR (**b**) among T2D individuals

The GRADE assessment for each outcome, summarized in Supplemental Table 3, revealed 'low' grades for acute PPGR in normoglycemic and T2DM, mainly downgraded due to inconsistency and indirectness of these outcomes. Evidence on long-term parameters fasting glucose, HbA_{1c} were graded as 'very low' due to low ratings for consistency, directness, and precision that led to decrease in the level of certainty.

Discussion

In this systematic review and meta-analysis, we found that pulse intake enhances glycemic regulation on both acute post-prandial responses and long-term glycemic indices. We demonstrate that pulse intake leads to clinically significant reductions in PPGRs, with a mean reduction of PPGR > 1 mmol/L in normoglycemic individuals, and > 2.5 mmol/L in those with T2D, and consequently significantly reduced insulin was observed ≥ 20 mIU/L. Long-term pulse intake was reported to reduce fasting glucose, HbA_{1c} and HOMA-IR with more pronounced effect in adults with T2DM.

Post-prandial glycemic control plays a crucial role in prevention of chronic diseases such as cardiovascular disease, in both normoglycemic and T2D individuals [88]. The estimated magnitude of the reduction in PPGR is similar to the reported effect of some glucose lowering therapies such as DPP-4 inhibitors [89, 90]. However, the certainty of evidence is impaired due to substantial inter-study variances. Possible modifiers were identified in acute RCTs, such as differences in pulse type, processing methods, and the control used as a comparison, which were explored by subgroup analysis. Although lentils are suggested by subgroup analysis to be the most potent type in controlling PPGR, other types of pulses still show a clinically significant impact (range −1.60 to −0.95 mmol/L) in normoglycemic adults with substantial inter-study heterogeneity. There are only a few trials that have assessed the impact of processing on post-prandial glycemic responses and the results are mixed with some RCTs finding no significant impact of processing in attenuating PPGR, while others suggest that pulse flour resulted in significantly higher PPGR in comparison to other physical forms [6, 18, 20]. Our meta-analysis supports the finding that intervention foods using pulse flour were found to be 50% less effective in attenuating PPGR when comparing to other physical forms. However, pulse flour used as intervention in the RCTs was incorporated into bakery products or pasta, with the flour being only 25–35% of the composition of final product; the incorporation of legume flour with cereal flours resulted in a lower effect when compared to whole pulses which were mostly consumed alone. Nevertheless, the lower efficacy of pulse flour could also be explained by breakage of the cell walls during the milling process, resulting in increased exposure of the starch to digestive enzymes whilst wet pureeing may result in cell separation, keeping more cells intact [6]. However, due to the high heterogeneity within subgroups, possibly due to the presence of different pulse types within a subgroup and lack of standardized protocol for food processing, definitive outcomes cannot be concluded and, therefore, more studies are

needed to investigate effects of processing on post-prandial glycemic handling.

In alignment with blood glucose, pulse intake favorably affected post-prandial insulin levels with a larger effect in T2D population where reduction in PPGR was greater. There were large variations between RCTs with regards to characteristics of participants such as mean age (22–66 y) and BMI (20–31), that might influence insulin secretion and sensitivity.

Long-term RCTs show that pulse intake leads to a favorable impact on fasting blood glucose in adults with and without T2D, and improved HbA_{1c} and HOMA-IR in those with T2D. The attenuation of fasting blood glucose was small in normoglycemic individuals (mean difference of ~0.06 mmol/L over median duration of 6 weeks), and greater in with T2D (mean difference of ~0.5 mmol/L over median duration of 8 weeks). We conducted a comparison of ES considering presence of diabetes as a modifier, and found significant differences between both conditions ($p < 0.05$) (Supplemental Fig. 7).

Post hoc meta-regression was performed to investigate the effect of pulse dose and study duration, and found low doses of pulses were more effective in reducing fasting blood glucose in adults without T2DM. However, there was no significant effect of study duration in modifying the ES (Supplemental Figs. 8 and 9). Our findings are in agreement with Sievenpiper et al. reporting inverse association between pulse dose in interventions and ES [35].

The reduction of HbA_{1c} (mean reduction of ~ 0.3%) is also considered to be clinically significant as the effect is comparable to low doses of some oral anti-diabetic agents such as α-glucosidase inhibitors [91]. Considering that HbA_{1c} reflects average glucose levels over the 8–12-week life span of erythrocytes [92], it is not surprising that some studies with an intervention duration shorter than this did not report an improvement in this measure. This together with subgroup analysis of study duration emphasizes the importance of conducting long-term RCTs of > 8 weeks in duration to report the outcomes of pulse intake and other dietary interventions on measures of glycemic control.

The beneficial effect of pulse intake on regulation of glucose metabolism could be related to several mechanisms. The bioavailability of carbohydrates from pulses can be reduced by factors such as low free sugar content and high levels of resistant starch [13]. In cooked whole or blended pulses, the presence of thick cell walls is likely to prevent access of amylolytic enzymes to the starch substrate [93]. Thermal processing increases fiber solubility, but the impact of this on glycemic effects is not known [94]. Furthermore, the crystalline nature of pulse starch and presence of fiber polysaccharides (both soluble and insoluble) as well as protein and lipids, contribute to delaying the gastric transit thereby slowing the arrival of food into the small intestine and hence lower the glycemic response [13, 95].

Other systemic effects may be via the microbial fermentation of fiber and resistant starch in the colon to short-chain fatty acids (SCFA) such as propionate, butyrate and acetate [96]. These SCFA reduce glucose release from the liver and thus promote muscle glycolysis, improved insulin secretion and glucose homeostasis via gut-brain axis and suppression of free fatty acid synthesis [97]. The soluble fiber is suggested to have beneficial impact on reduction of post-prandial glycemic effects attributing to the viscosity and gel-forming properties [98, 99]. Presence of fiber along with slowly digestible starch in pulses has been linked to improved blood glucose profile, insulin sensitivity and urinary C-peptide, and tends to normalize insulin levels in individuals with hyperinsulinemia [100].

To our knowledge, this is the first meta-analysis summarizing the impact of pulse intake on acute PPGR reported after pulse intake, and the most comprehensively assessing long-term impact of pulse consumption on glycemic handling indices. Post-prandial glycemic biomarkers are highly correlated with long-term indices and are considered as independent risk factors in progression of several health conditions such as diabetes and coronary heart diseases [101]. Therefore, including acute post-prandial trials in this review, and adopting raw mean difference over standardized mean difference beside employment of meta-regression allow better understanding over previous meta-analysis regarding the role of pulses in controlling glycemic indices [35]. Furthermore, we have assessed the certainty of the evidence by following GRADE method, and calculated the prediction intervals to estimate clinical consequences of the heterogeneity and to provide a range into which we can predict the outcome of future studies to fall based on current evidence. Our prediction intervals are broad including both positive and negative intervals, reducing the confidence in predicting that results of a future trial would favor pulse intake, although broad prediction intervals are common in RCTs. However, there are several limitations in our analysis that should be considered. First, the risk of bias ranged from 'some concerns' to 'high risk', and the quality of evidence was graded from 'low to 'very low'. This is largely due to substantial inter-study heterogeneity that remained unexplained despite subgroup analysis. Additional variables such as ethnic background, genetic predisposition, physiological factors such as age, gender and BMI, lifestyle of the participants might contribute toward observed heterogeneity in reported outcomes and thus affecting the grading of the evidence. The quality of the RCTs was downgraded mostly due to inappropriate way in conducting or reporting of randomization process, or due to unavailability of trial protocol or register information. These factors collectively reinforce importance of high quality RCTs to support the beneficial effect of pulse intake

on glycemic handling [102]. Second, we have included only RCTs with defined pulse consumption in the meta-analysis while excluding those that included pulses in selective eating patterns such as low GI or high-fiber diets. While this may have reduced the number of studies included, it also increased knowledge about particular types and forms of pulses. Third, there were 28 studies excluded due to inability of accessing the full text, and 3 papers were excluded as they were not available in English language. These collectively might have resulted in publication bias. Finally, the data extraction procedure was performed by single author which might introduced some biases.

Overall, pulse intake significantly reduced PPGR in both normoglycemic and individuals with T2D and, therefore, are recommended for consumption as a low GI food. Long-term pulse consumption resulted in favorable effects on measures of glycemic control especially in those with T2D. Although whole or pureed lentils showed more promising effects, due to high heterogeneity between studies, it is not possible to give a specific recommendation with regards to pulse type, dose, form (i.e. processing method) and duration of intake. Carefully controlled acute studies are required to study the impact of differently processed pulses on glycemic parameters. Furthermore, well-designed long-term RCTs are needed to establish effectiveness of pulse rich diets and dose–response relationships to refine dietary recommendations for pulse intake.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00394-021-02685-y.

Author contributions MSH, CO, MDC and CB designed the protocol of the systematic review. MSH and LLO screened the databases, identified the included RCTs independently and assessed risk of bias. MSH extracted and MSH and MH analyzed the data. All authors contributed toward manuscript writing, read and approved the final version. None of the authors had a conflict of interest.

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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